REMARKS

Claims 69-94 are now pending. Favorable reconsideration is respectfully requested.

The present invention relates to a mammalian non-human female animal having a complete depletion of ovarian primordial follicles and at least one characteristic selected from the group consisting of depletion of ovarian follicles, irregular ovarian cyclicity, cessation of estrous cyclicity, elevated FSH levels, erratic ovarian 17β-estradiol levels, loss of bone mineral density, and reduced ovarian weight. See Claim 69.

The present invention also relates to a method of inducing ovarian failure in a mammalian non-human female animal other than a mouse or a rat, comprising administering to the animal 4-vinylcyclohexene diepoxide at a dosage of at least 100 mg/kg/day. See Claim 86.

The present invention also relates to a method of controlling the size of a mammalian non-human animal population, comprising administering 4-vinylcyclohexene diepoxide at a dosage of at least 100 mg/kg/day to cause at least partial ovarian failure in at least a portion of the female members of the animal population. See Claim 91.

The rejection of the claims under 35 U.S.C. §102(b) over Kao et al. as evidenced by Amant et al. and Osei-Hyiaman et al. set forth in paragraph 4) at page 4 of the Office Action is respectfully traversed. Kao et al. fail to describe the claimed animal.

Kao et al. describe an investigation into the effects of 4-vinylcyclohexene diepoxide (VCD) as an ovatoxin in rats and mice. As explained in the second column of page 67, VCD is an industrial chemical that may cause undesirable effects on women exposed thereto. Those animals were administered VCD at a dose of 80 mg/kg/day.

As described by Kao et al., the largest level of depletion of ovarian primordial follicles was 43%. Therefore, the reference fails to disclose an animal having a complete

depletion of ovarian primordial follicles as claimed. Kao et al. also fail to disclose the mice or rats described therein have any of the characteristics recited in Claim 69.

While Amant may describe that 17β-estradiol levels are erratic and then diminish through perimenopause and menopause, there is no evidence that the animals described in Kao et al. have erratic levels of 17β-estradiol or are in perimenopause or menopause.

While Osei-Hyiaman et al. may state that a decrease in bone mineral density is associated with menopause, there is no evidence that the animals described in Kao et al. have a decrease in bone mineral density or are in menopause.

In view of the foregoing, Kao et al. fail to disclose the claimed animal. Accordingly, the pending claims are not anticipated by that reference. Therefore, withdrawal of this ground of rejection is respectfully requested.

The rejections of the claims under 35 U.S.C. §103(a) over Kao et al. in view Abel et al., Judd and Mulholland et al. as set forth at pages 7-10 of the Office Action are respectfully traversed.

As discussed above, Kao et al. fail to disclose an animal having a complete depletion of ovarian primordial follicles as claimed. Kao et al. also fail to disclose the mice or rats described therein have any of the characteristics recited in Claim 69.

There is no motivation to modify Kao et al. to arrive at the claimed animal. First, there is no suggestion in Kao et al. or any of the cited secondary references that the claimed animal could be obtained.

Abel et al. describe the effects of hormone replacement therapy in a primate model of menopause. See the Abstract. The primates described therein were obtained by ovarectomy.

Judd is a review article that describes the hormonal dynamics associated with menopause. See the Abstract.

Mulholland et al. the effect of hormonal deprivation on the bladder defense mechanism. See the Abstract.

There is no suggestion from the references that a non-human female animal with complete depletion of ovarian primordial follicles and at least one characteristic selected from the group consisting of depletion of ovarian follicles, irregular ovarian cyclicity, cessation of estrous cyclicity, elevated FSH levels, erratic ovarian 17β-estradiol levels, loss of bone mineral density, and reduced ovarian weight could be produced. There is simply no suggestion in the cited references that the animals described by Kao et al. could be modified successfully to arrive at the animal claimed in the present invention.

In view of the foregoing, the claimed animal is not suggested by the combination of Kao et al., Abel et al., Judd and Mulholland et al. Accordingly, the claims are not obvious over those references.

The rejection of Claims 37 and 40 under 35 U.S.C. §112, second paragraph, is believed to be obviated by the amendments submitted above in part and is, in part, respectfully traversed.

Claims 37 and 40 correspond to newly-added Claims 86 and 92, respectively.

The object of Claim 86 is to induce ovarian failure in the animal specified in the claim. Administering VCD as specified in the claim accomplishes that object.

The object of Claim 92 is to control the size of an animal population specified in that claim. As discussed in the specification, administering VCD at the specified dose has detrimental effects on the reproductive ability of females. The method of Claim 92 is based on this discovery. Administering VCD as specified in the claim to the female members of the animal population will result in fewer offspring, thereby controlling the size of the population. Therefore, administering VCD as claimed accomplishes the object of the claim.

In view of the foregoing, the claims are definite within the meaning of 35 U.S.C. §112, second paragraph. Withdrawal of this ground of rejection is respectfully requested.

A copy of reference AAJ submitted with the Information Disclosure Statement on June 7, 2004 has been submitted for the Examiner's consideration.

Applicants submit that the present application is in condition for allowance. Early notice to this effect is earnestly solicited.

Respectfully submitted,

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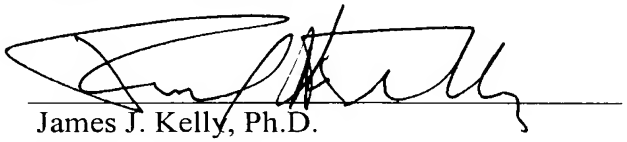

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